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STUDIES IN A CASE OF ACUTE BICHLORIDE OF MERCURY POISONING TREATED BY THE NEWER METHODS, AND FOLLOWED BY RECOVERY.

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(From the Laboratory of Dr. Jacob Rosenbloom.)

1. Introduction and Review of the Literature. In recent years the treatment of the formerly hopeless cases of bichloride of mercury poisoning has developed along new lines that seem to be of great promise as regards the recovery of these cases.

Lieb and Goodwin[1] emphasized the importance of repeated gastric lavage to remove the mercury excreted into the stomach and Lambert and Patterson[2] have shown the necessity of utilizing every means we have for the purpose of elimination of the mercury from the body. They have shown the necessity of frequent gastric lavage and colon irrigations continued until the washings are free from mercury when tested chemically. Other workers have advised procedures some of which when studied experimentally have failed to protect against mercury poisoning.

Hall[3] recommended the use of a solution of potassium iodide, quinine hydrochloride, hydrochloric acid and water, but Barbour[4] and also Sansum[5] have shown that this antidote is valueless. Fantus[6] has shown that the use of egg albumen is without value unless

[1] Jour. Am. Med. Assn., 1915, lxiv, 2041.
[2] Arch. Int. Med., 1915, xvi, 865.
[3] Jour. Am. Pharm. Assn., 1915, iii, 182.
[4] Jour. Am. Med. Assn., 1915, lxiv, 736.
[5] Ibid., 1918, lxx, 824.
[6] Jour. Lab. and Clin. Med., 1916, i, 879; 1917, ii, 813.

it is given along with or immediately after the poison. He considers lavage with a solution of sodium acetate to be of more value. He also recommends the use of Carter's antidote, a tablet containing 0.36 gram of sodium phosphite and 0.24 gram of sodium acetate. If this is not available he uses a mixture of 5 parts of hydrogen peroxide, 10 parts of water and 1 gram of sodium hypophosphite, using ten times as much hypophosphite as poison and this is followed by copious lavage with the diluted reagent. He also found that sodium bicarbonate had antidotal power, and he uses it in amounts of twenty-five to fifty times the amount of poison taken.

Sabbatani[7] has mentioned the possible value of sulphur, hydrogen sulphide or sodium thiosulphate solutions, as he thinks the use of them as gargles, enemas and hypodermically combat the stomatitis and colitis and impede absorption of the mercury Linhart[8] advises the use of sodium phosphite as an antidote. Wilms[9] considered that the use of calcium sulphide given by mouth and intravenously was of antidotal power in the treatment of these cases. Hayward and Allen[10] also suggested the use of calcium sulphide. Evans[11] a long time ago considered alkalies of use in the treatment of bichloride of mercury poisoning, and recently Hirsch[12] has suggested the use of sodium bicarbonate as an antidote. Burmeister[13] has emphasized the value of repeated venesections and transfusions in the treatment of these cases. Turrettini[14] reports favorably the use of intravenous injections of glucose solution in mercury poisoning. Carter[15] thinks he has successfully treated these cases by giving by mouth a combination of sodium phosphite and sodium acetate to reduce the mercuric salt to mercurous. Sansum[16] has shown experimentally that this method is without value, and Haskell and Courtney[17] have shown experimentally that the intravenous injection of calcium sulphide is too dangerous a procedure to be of value and that its effect is due to the fluid introduced. Schisler[18] has advised the use of magnesium oxide in these cases. Milian and Saint-Avid[19] advised the use of intravenous injections of alkali and

[7] Biochem. Centralbl., 1906, v, 502; Riforma Med., 1907, xxiii, 767; Arch. Int. de Pharmacol., 1907, xvii, 319.
[8] New York Med. Jour., 1913, xcvii, 1236; Jour. Lab. and Clin. Med., 1917, ii, 722.
[9] Jour. Lab. and Clin. Med., 1917, ii, 445; Lancet-Clinic, 1915, cxiv, 555; 1916, cxv, 460; Ther. Digest, 1916, xi, 71.
[10] Jour. Am. Med. Assn., 1913, lx, 1827.
[11] Med. and Phys. Jour., London, 1800, iii, 535.
[12] Chicago Med. Rec., 1914, xxxvi, 444.
[13] Jour. Lab. and Clin. Med., 1917, ii, 500.
[14] Rev. Med. de la Suisse romande, 1915, xxxv, 204.
[15] Chicago Med. Record, 1914, xxxvi, 444; Critic and Guide, 1915, xviii, 266; Am. Jour. Clin. Med., 1914, xxi, 314.
[16] Jour. Am. Med. Assn., 1918, lxx, 824.
[17] Jour. Lab. and Clin. Med., 1917, iii, 110.
[18] Jour. Mississippi Med. Assn., 1917, xiv, 173.
[19] Paris méd., 1917, vii, 213.

glucose. Holm[20] advised the use of calcium sulphide. Macnider,[21] from his studies on the protection that alkalies give to the development of tubular nephritis in uranium poisoning, advises the use of alkalies in acute mercury poisoning. He found that death in acute bichloride of mercury poisoning in dogs was due to shock associated with a mercury enteritis and to delayed kidney damage associated with acidosis. He thought that the delayed kidney injury was not due to the action of mercury as such during its elimination by this organ. Anderson[22] advised water irrigations through a cecostomy wound to hasten the bowel elimination in these cases. Weiss[23] has published a method for treating these cases somewhat similar to that described by Lambert and Patterson, with the exception that the frequent gastric and colonic lavage is omitted. One improvement in his method over that of Lambert and Patterson is that the urine is kept alkaline to a saturated solution of methyl red in alcohol, on the basis that Fischer has shown that if the urine of a nephritic cannot be made alkaline to methyl red this patient continues in a dangerous state.

There is no doubt that the treatment of cases of acute bichloride of mercury poisoning by the newer methods has produced better results than by the former treatment, which was really watching the case until fatal anuria developed. Schisler[24] reports 16 cases with 5 deaths, dose from 3 to 170 grains. Weiss[25] reports 28 cases with 1 death, dose from 3 to 82 grains, the cases being treated by the newer methods.

Brown and Baskett,[26] Lewis and Rivers,[27] and Cohen and Bernhard[28] have reported cases with recovery following the Lambert-Patterson treatment. Nolan[29] has recently presented a very able review of recent work in regard to acute mercuric chloride poisoning. It should be recalled that in the elaboration of any method of treatment of bichloride of mercury poisoning the following conditions are to be met:

The development of anuria, which occurs in from twelve hours up to ten days after taking the poison, due to an acute nephritis and enteritis, and the colitis, due to the inflammation caused by excretion of the mercury into the intestine. Together with this there exists an acid intoxication, and, as Nolan well says: "The therapeutic problems are (1) to reduce the toxicity of the mercury

[20] Jour. Michigan State Med. Soc., 1917, xvi, 270.
[21] Jour. Med. Research, 1912, xxi, 79; Jour. Exper. Med., 1918, xxvii, 519; 1916, xxiii, 171.
[22] Surg., Gynec. and Obst., 1915, xx, 350; Harrold: Ann. Surg., 1916, lxiii, 127.
[23] Jour. Am. Med. Assn., 1917, lxviii, 1618.
[24] Jour. Mississippi Med. Assn., 1917, xiv, 173.
[25] Ohio State Med. Jour., 1917, xiii, 595; Jour. Am. Med. Assn., 1917, lxviii, 1618.
[26] Jour. Am. Med. Assn., 1917, lxviii, 1622.
[27] Johns Hopkins Hosp. Bull., 1916, xxvii, 193.
[28] Jour. Am. Med. Assn., 1916, lxvii, 1019.
[29] Interstate Med. Jour., 1917, xxiv, 997.

in the system; (2) if possible to hasten its elimination; (3) to combat the acid intoxication and its attendant evils.

That some method to facilitate the elimination of the mercury present in the body is needed is shown by the following data. In a case of bichloride of mercury poisoning in which death followed ten days after taking the drug I[30] was able to find the following amounts of mercury still present in the body:

Organ.	Mercury present in milligrams.
Kidney	5.8
Spleen	1.7
Liver	39.82
Brain	1.08
Stomach	1.50
Small intestines	4.80
Large intestines	7.28
Heart	5.81
Lungs	trace
Blood	52.2
Muscle	25.8
Bile	trace
Stomach contents	12.2
Intestinal contents	8.6
Rectal contents	8.4

Some recent experimental work of Sansum[31] seems to show that when an amount of mercuric chloride in excess of 4 mg. has entered the tissues at large, death regularly occurs, and that we have no adequate grounds for believing that death is preventable by any known form of treatment. He thinks that therapeutic efforts should be directed toward the mechanical removal of the poison from the lumen of the alimentary tract and antidoting the poison before it leaves the portal circuit—that is, particularly before absorption. However, Sansum does not take into consideration the treatment of the acidosis present in these cases.

2. CASE HISTORY. A man, aged thirty-five years, physically well but mentally sick, chewed up three 7½-grain tablets of bichloride of mercury at twelve o'clock noon. He vomited at two o'clock, and the vomitus contained streaks of blood. He was given, at three o'clock, the whites of three eggs and a quart of milk, and this was vomited in ten minutes. The treatment outlined in this paper was started at seven o'clock—that is, seven hours after the poison was taken. During the first two days the bowels moved every two or three hours and the movements contained shreds of mucus and blood. The stomach washings also showed clumps of clotted blood during this time; a severe stomatitis developed on the third day. Excruciating lumbar pain came on during the second day and lasted for three days. At the end of the first week I felt confident that the patient would recover. He was carefully watched for one month

[30] Rosenbloom: Jour. Biol. Chem., 1915, xx, 123.
[31] Jour. Am. Med. Assn., 1918, lxx, 824.

and was discharged as completely recovered. He is at present (twelve months after his illness) well in all respects and no trace of damage to his kidneys can be found by examining his urine or by testing the kidneys by the functional methods.

The following tables contain the data obtained in the study of this case of acute bichloride of mercury poisoning:

TABLE I.—ANALYSES OF BLOOD.

Day.	Total non-protein nitrogen. Mgm. per 100 c.c. blood.	Urea nitrogen. Mgm. per 100 c.c. blood.
1	42	20
2	48	25
3	84	66
5	140	94
8	172	140
10	176	138
12	141	100
14	92	70
17	60	40
20	48	25
22	40	20
24	36	18
25	32	12
28	28	11
30	23	10

3. LABORATORY STUDIES. Foster,[32] Phillip,[33] Myers and Lough,[34] Squier and Myers,[35] Campbell and Hunter,[36] Woods,[37] and Lewis and Rivers[38] have published chemical studies of the blood in bichloride of mercury cases. When my results are compared with those obtained by these observers it may be noted that at no time did the blood non-protein nitrogen or urea-nitrogen reach the high values found by some of these workers. I consider that the lower values obtained in this case were due to the better excretion by the kidney, owing to the treatment used. However, there is a marked increase in the non-protein nitrogen and the urea-nitrogen of the blood in acute bichloride of mercury poisoning.

DISCUSSION OF TABLE II. The presence of large amounts of acetone and diacetic acid in the urine with the high ammonia nitrogen points to the existence of a definite acidosis. That an acidosis exists in uremia was shown long ago by von Jaksch.[39] Straub and Schlayer,[40] Sellards,[41] Palmer and Henderson,[42] How-

[32] Arch. Int. Med., 1915, xv, 755.
[33] Med. Klinik, 1913, ix, 912.
[34] Arch. Int. Med., 1915, xvi, 536.
[35] Jour. Urol., 1918, ii, 1.
[36] Jour. Biol. Chem., 1917, xxxii, 195; 1918, xxxiii, 169; Arch. Int. Med., 1917, xx, 919.
[37] Arch. Int. Med., 1915, xvi, 577.
[38] Johns Hopkins Hosp. Bull., 1916, xxvii, 193.
[39] Ztschr. f. klin. Med., 1889, xiii, 350.
[40] München. med. Wchnschr., 1912, lix, 569.
[41] Johns Hopkins Hosp. Bull., 1914, xxv, 141.
[42] Arch. Int. Med., 1915, xvi, 109.

land and Marriott[43] and Peabody[44] have also found an acidosis present in uremia. However, this case shows that there was an overproduction of acid bodies and that the acidosis was not entirely due to an imperfect excretion of the acid radicals, as is claimed by Palmer and Henderson. The existence of this acidosis shows the necessity of treating cases of bichloride of mercury poisoning with this factor in mind. It shows the necessity of using the low protein, low fat and high carbohydrate diet and the use of alkalies to keep the urine persistently alkaline to methyl red. It may be noted that on the twenty-fourth day of the illness all damage to the kidney had been repaired, as evidenced by changes in the urine. From this day on the urine was normal in all respects. The acidosis was kept under control by the use of alkalies, glucose and high carbohydrate, low protein and low fat diet. I think that the control of the acidosis was an important feature in the successful outcome of this case.

TABLE II.—ANALYSES OF THE URINE.

Day	Volume, c.c.	Sp. gr.	Reaction.	Protein gm. per liter.	Nitrogen gm.	Ammonia nitrogen. Gm.	Ammonia nitrogen. Per cent. of total Nitrogen.	Acetone.	Diacetic acid.	Microscopic. Hyaline casts.	Granular casts.	Red blood cells	White blood cells	Epithelial cells.
1	300	1010	Acid	3.5	3.8	0.22	6.0	xxxx	xxxx	xx	xx	xx	xx	xxx
2	420	1008	Acid	4.0	3.9	0.23	5.8	xxxx	xxxx	xx	xx	xx	xx	xx
3	880	1009	Acid	4.0	5.2	0.53	10.2	xxxx	xxxx	xx	xx	xx	x	xx
4	1600	1010	Acid	1.0	6.2	0.90	14.5	xxxx	xxxx	xx	xx	xx	x	xx
5	3000	1012	Acid	1.0	7.1	0.89	12.6	xxxx	xxxx	xx	xx	xx	x	xx
6	4200	1010	Alkaline	0.5	8.1	0.73	8.9	xxxx	xxxx	xx	xx	xx	x	xx
7	4800	1010	"	0.5	7.2	0.91	12.7	xxx	xxx	xx	xx	xx	x	xx
8	6000	1010	"	0.5	10.4	1.00	9.6	xxx	xxx	x	x	xx	x	xx
9	6400	1012	"	Trace	11.6	1.03	8.9	xx	xx	x	x	xx	x	x
10	5200	1010	"	"	10.8	0.93	8.6	xx	xx	x	x	xx	0	x
11	5000	1009	"	"	9.7	0.78	8.1	xx	xx	x	x	x	0	x
12	4840	1010	"	0.5	10.2	0.82	8.0	xx	xx	x	x	x	0	x
13	3200	1012	"	0.5	10.3	0.77	7.5	x	xx	x	x	x	0	x
14	3000	1014	"	Trace	9.8	0.72	7.4	x	x	x	x	x	0	x
15	1500	1015	"	"	9.4	0.61	6.5	x	x	x	x	x	0	x
16	1420	1018	"	"	8.6	0.53	6.2	x	x	x	x	x	0	x
17	1400	1016	"	"	7.2	0.44	6.1	0	0	x	x	x	0	0
18	1200	1015	"	"	6.8	0.35	5.1	0	0	x	x	x	0	0
19	1100	1010	"	"	7.4	0.39	5.3	0	0	x	x	x	0	0
20	1000	1018	"	"	7.2	0.36	5.0	0	0	x	x	x	0	0
21	1800	1012	"	"	7.0	0.41	5.8	0	0	x	x	x	0	0
22	1500	1012	"	0	6.8	0.32	4.7	0	0	x	x	x	0	0
23	1450	1014	"	0	5.6	0.25	4.5	0	0	x	x	x	0	0
24	1860	1010	"	0	5.8	0.29	5.0	0	0	0	0	0	0	0
25	1010	1016	"	0	7.2	0.37	5.1	0	0	0	0	0	0	0
26	1200	1015	Acid	0	7.1	0.37	5.2	0	0	0	0	0	0	0
27	1280	1018	"	0	8.6	0.55	4.6	0	0	0	0	0	0	0
28	1400	1017	"	0	8.4	0.40	4.8	0	0	0	0	0	0	0
29	1030	1014	"	0	7.6	0.34	4.5	0	0	0	0	0	0	0
30	1200	1018	"	0	7.2	0.32	4.4	0	0	0	0	0	0	0

[43] Johns Hopkins Hosp. Bull., 1916, xxvii, 63.
[44] Arch. Int. Med., 1914, xiv, 236; 1915, xvi, 955.

TABLE III.—PHENOLSULPHONEPHTHALEIN TEST.

Day.	Per cent. of dye excreted in the First hour.	Second hour.	Total.
1	trace	trace	
2	trace	trace	
3	5	5	10
5	10	5	15
8	15	10	25
10	18	12	30
12	20	15	35
14	18	18	36
17	20	18	38
20	25	20	45
22	30	20	50
24	35	20	55
25	35	15	50
28	40	20	60
30	40	20	60

Discussion of Table III. This table shows very nicely the damage done to the kidneys by the bichloride of mercury and the gradual resumption of the kidney function in the later days of the illness. From the merest trace of the dye being excreted on the first and second days following the ingestion of the mercury, the amount gradually increased until on the twenty-second day it could almost be considered normal and on the twenty-eighth day the excretion of the dye was normal.

TABLE IV.—STUDIES OF THE MERCURY EXCRETED.

Day.	Urine.	Mercury present in: Colon washings.	Gastric washings.
1	xxxx	xxxx	xxxx
2	xxx	xxx	xxx
3	xxx	xxx	xxx
5	xxx	xxx	xxx
8	xxx	xxx	xxx
10	xxx	xx	xx
12	xx	x	x
14	x	x	x
17	x	x	x
20	x	x	x
22	Trace	Faint trace	Faint trace
24	Faint	"	"
25	"	Very faint	Absent
28	Absent	Absent	"
30	"	"	"

Discussion of Table IV. The necessity of continuing the treatment of these cases for a long period of time is clearly shown by a study of this table. Fairly distinct amounts of mercury were still present in the urine, colon washings and gastric washings for a period of twenty days following the ingestion of the mercury. Faint traces were still present up until the twenty-eighth day. There is no doubt that the best method is to continue the treatment used in these cases until the urine, the colon washings and the gastric washings are free from mercury.

The method of treatment that was used in this case was a combination of various procedures that have been advised for the treatment of mercury poisoning. It may be tabulated as follows: In this case the sodium phosphite and sodium acetate were not used, instead the hypophosphite-peroxide mixture was used.

Method of Treatment to Be Used in Cases of Bichloride of Mercury Poisoning.

1. Administer the whites of three eggs beaten up in a quart of milk and then empty the stomach by siphonage.

2. Give 300 c.c. of fresh calcium sulphide solution, containing 1 grain to 1 ounce of water by mouth.

3. Wash out the stomach with fresh calcium sulphide solution, 1 grain to 1 ounce of water.

4. Administer in powder or tablet 0.36 gram of sodium phosphite and 0.24 gram of sodium acetate. If this is not available give the following:

Sodium hypophosphite	1 gram
Water	10 mils.
Hydrogen peroxide	5 mils.

Use ten times as much of the hypophosphite as poison taken. Give a copious lavage of stomach with the above antidote diluted twenty times. Give the above undiluted antidote every eight hours for two days.

5. Pour through the stomach tube after the above lavage a solution of 3 ounces of sodium sulphate and 6 ounces of water containing 5 grains of calcium sulphide. Let these solutions remain in the stomach.

6. Give intravenously after withdrawing 600 c.c. of blood, 800 c.c. of Fischer's solution or of bicarbonate-glucose solution.

7. Wash out the stomach morning and night, giving by the mouth after each washing, 5 grains of calcium sulphide dissolved in 3 ounces of water. Continue this lavage until the stomach washings are free from mercury when tested by Elliott's[45] method and until the urine is free from mercury.

8. Give high colon irrigations of warm water morning and night, using 8 gallons of the water for each treatment.

9. Give a hot pack twice daily.

10. Give 8 ounces of milk every second hour.

11. Give every second hour 8 ounces of the following solution, by mouth, alternating with the milk:

Potassium bitartrate	dr. j
Sodium citrate	dr. j
Sucrose	dr. j
Lactose	dr. iv
Lemon juice	oz. j
Boiled water	oz. xvj

[45] Jour. Am. Med. Assn., 1917, lxviii, 1693.

12. Force the patient to drink large quantities of the alkaline waters, such as Celestin's Vichy or Kalak water.

13. Give a low fat and low protein and high carbohydrate diet for four weeks. Avoid salt in food, as it increases the absorption of the mercury.

14. Give by continuous proctoclysis a solution containing 1 dram potassium acetate, 4 drams glucose and 3 drams sodium bicarbonate to the pint.

15. Keep the urine alkaline to methyl red.

16. Continue rest of treatment until recovery, usually a period of three weeks.

A CASE OF BULIMIA, WITH REMARKS ON THE CAUSAL TREATMENT OF SOME FUNCTIONAL DISEASES.[1]

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Though bulimia is always mentioned in the proper connection, it rarely gets into indexes, and does not seem to be very frequent in practice. The descriptions in text-books are as a rule very good, but the tendency to place bulimia with polyphagia in diabetes or exophthalmic goitre must raise doubt in the mind of anyone who sees a case like the one I report, or even one who reads a current definition, as to its place in pathology. Recent studies of hunger, especially those of Cannon and Carlson and their collaborators, give an added interest to all anomalies of hunger. For these reasons and on account of the outcome of the case, I report the history of a patient sent me recently for investigation.

G. S., aged thirty-six years, architect.

Diagnosis. Bulimia, pansinusitis, acute rhinitis, pharyngitis and laryngotracheitis, caries of teeth, alveolar abscess.

Complaint. The patient has to eat more frequently and in larger quantities than normal. Hunger attacks begin with general feeling of uneasiness, followed, if food is not taken, by an intense headache, generally in the eyes or frontal region. Sometimes headache comes quickly if food is not taken, sometimes it is delayed. The patient eats from five to fifteen times in twenty-four hours. More accurately, he has no regular meal time and does not always eat with the family, but eats at any time he feels a craving. At family meals he may take a bowl of soup, crackers, rice or bread. He has acquired the habit of eating toasted bread cubes, which he always has with him in large tin cans. He will eat a bowlful of these, or five to eight shredded wheat biscuits each time he gets hungry.

[1] Read before the Association of American Physicians, Atlantic City, May 7, 1918.